DISSERTATION ON

A STUDY ON

ACID – BASE STATUS AMONG INTENSIVE MEDICAL CARE UNIT PATIENTS

Submitted in partial fulfillment of

Requirements for

M.D.DEGREE BRANCH I INTERNAL MEDICINE

Of

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CERTIFICATE

This is to certify that this dissertation entitled **" A STUDY ON ACID BASE STATUS AMONG INTENSIVE MEDICAL CARE UNIT PATIENTS "** submitted by **Dr. SASIREKHA K** appearing for Part I & II M.D Branch I Internal Medicine Degree examination in March 2008 is a bonafide record of work done by her under by direct guidance and supervision in partial fulfillment of regulations of the TamilNadu Dr.M.G.R. Medical University, Chennai. I forward this to the TamilNadu Dr.M.G.R. Medical University, Chennai, TamilNadu, India.

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DECLARATION

I solemnly declare that dissertation entitled **" A STUDY ON ACID BASE STATUS AMONG INTENSIVE MEDICAL CARE UNIT PATIENTS "** is done by me at Madras Medical College & Government General Hospital, Chennai, during 2006 – 2007 under the guidance and supervision of Prof.V.K.Rajamani, M.D.

The dissertation is submitted to The TamilNadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree (Branch I) in Internal Medicine.

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CONTENTS

Introduction

INTRODUCTION

A little learning is a dangerous thing.

Drink deep, or taste not the pyrean spring

- ***Alexander Pope***

Acid Base abnormalities are common in critically ill patients. Our ability to describe acid base disorders must be precise. Small differences in corrections for anion gap, different types of analytical processes, and the basic approach used to diagnose acid base aberrations can lead to markedly different interpretation and treatment strategies for the same disorder (1).

Life is a struggle;

Not against sin;

Not against money power;

Not against malicious animal magnetism;

But against [H^+] ions

- ***H.L. Mencken***

Mencken was neither a physiologist nor a physician but he knew the importance of [H^+] ions.

To maintain homeostasis the body has to keep [H^+] ions concentration at 40 nanomoles/L (2). A blood pH less than normal (normal range 7.35 – 7.45) is called acidemia; the underlying process causing acidemia is called

acidosis. Similarly alkalemia and alkalosis refer to the pH and underlying process respectively. While an acidosis and an alkalosis may coexist, there can be only one resulting pH. Therefore acidemia and alkalemia are mutually exclusive conditions (3).

The metabolic and respiratory that regulate systemic pH are described by the Henderson – Hasselbach equation;

$$pH = 6.1 + \log (HCO_3 / PaCO_2 \times 0.03)$$

Alternatively H^+ ions can be expressed directly as (4).

$$H^+ = 24 \times (PCO_2 / HCO_3^-)$$

Primary change in $PaCO_2$ can cause acidosis or alkalosis, depending on whether $PaCo_2$ is above or below the normal value of 40 mm Hg. Primary alkalosis of PCo2 evokes cellular buffering and renal adaptation. A primary change in the plasma HCO_3^- as a result of metabolic or renal factors results in compensatory changes in ventilation that blunt the changes in blood pH. Such respiratory alterations are referred as secondary or compensatory changes, since they are occurring in response to primary metabolic changes.

Simple and mixed acid base disorders are commonly encountered in clinical practice and are particularly frequent in critically ill patients (5). Acid base disorders contribute importantly to patient morbidity and mortality, especially in critically ill (6). Therefore it is essential to recognize

and properly diagnose acid base disorders and understand their impact on organ function.

AIMS AND OBJECTIVES

- To analyze the acid base status among intensive care unit patients.
- To elicit various acid base disturbance in IMCU and toxicology units.
- To study the pattern of acid base disturbances.
- To study the effect of pH on the prognosis of the diseases.
- To elicit the causes for acid base disturbances.

REVIEW OF LITERATURE

Primary respiratory acid base disturbances invoke secondary metabolic response and primary metabolic acid base disturbance invoke secondary respiratory response (7).

SIMPLE ACID BASE DISORDERS

	Metabolic Acidosis	Metabolic Alkalosis	Respiratory Acidosis	Respiratory Alkalosis
Primary Changes	⇩ HCo_3^-	⇧ HCo_3^-	⇧ PCo_2^-	⇩ PCo_2^-
Compensation	⇩ PCo_2^-	⇧ PCo_2^-	⇧ HCo_3^-	⇩ HCo_3^-
Effect on pH	⇩ pH	⇧ pH	⇩ pH	⇧ pH

Since the definition of simple disturbances includes both the initial process producing a change in HCo_3^- and PCo_2^-, all the compensatory mechanisms affecting these substances, lack of appropriate compensation for a simple disturbance is an evidence for a mixed disturbance (8).

So it is critical to know both magnitude and the course of compensatory responses to simple disorders to identify the presence of mixed acid base disorders.

COMPENSATORY RESPONSE IN SIMPLE ACID BASE DISTURBANCES

Acid base disorders		Prediction of Compensation	Limits
Metabolic acidosis		PCo_2 = 1.5 x HCo_3^- + 8 Or PCo_2 will decrease by 1.25 mm Hg per mmol/L decrease in HCo_3^-	10 mm Hg
Metabolic alkalosis		PCo_2 = 0.9 x HCo_3^- + 16 Or PCo_2 will increase by 0.75 mm Hg per mmol/L increase in HCo_3^-	55 mm Hg
Respiratory alkalosis	Acute	HCo_3^- will decrease by 2 mmol/L per 10 mm Hg decrease in PCo_2	30 mEq/L
	Chronic	HCo_3^- will decrease by 4 mmol/L per 10 mm Hg decrease in PCo_2	45 mEq/L
Respiratory acidosis	Acute	HCo_3^- will increase by 1 mmol/L per 10 mm Hg increase in PCo_2	18 mEq/L
	Chronic	HCo_3^- will increase by 4 mmol/L per 10 mm Hg increase in PCo_2	15 mEq/L

A mixed acid base disturbance is defined as the simultaneous coexistence of two or more simple disorder in a same patient (9). Mixed acid base disturbances are more commonly predictable on the basis of clinical setting

and physical examination. Lab data mainly serve to confirm the clinical impression.

McCurdy et al (1981) suggested a systematic method to analyze acid base disorders by clinical approach (10).

I. Suspect the disturbances from history

II. Suspect the disturbances from physical examination

III. Evaluate routine lab data

1. HCo_3^-
    a) If increased think of metabolic alkalosis or compensated respiratory acidosis.
    b) If decreased think of metabolic acidosis or compensated respiratory alkalosis.
2. K^+
    a) If increased think of acidemia.
    b) If decreased think of alkalemia.
3. Cl^-
    c) If increased think of hyperchloremic metabolic acidosis
    d) If decreased think of metabolic alkalosis.
4. Anion Gap.

IV. Evaluate blood gas values to check appropriate compensation.

In the setting of known primary disorders the presence of normal pH implies a mixed disturbance, since compensation rarely corrects pH to normal. Generally more marked the primary disturbance, the less likely the pH will be normal, unless there is a mixed disorder.

Important Causes of mixed acid base disturbances :

1. Respiratory acidosis with metabolic acidosis

   Ex : Cardio pulmonary arrest

   Severe Pulmonary edema

2. Respiratory alkalosis with metabolic alkalosis

   Ex : Hepatic failure treated with diuretics

3. Respiratory alkalosis with metabolic acidosis

   Ex : Septic shock

4. Respiratory acidosis with metabolic alkalosis

   Ex : Corpulmonale treated with diuretics

5. Metabolic acidosis with metabolic alkalosis with respiratory alkalosis

   Ex : Diabetic keto acidosis with vomiting

6. Metabolic acidosis with metabolic alkalosis with respiratory acidosis

1) RESPIRATORY ACIDOSIS + METABOLIC ACIDOSIS :

This combination occurs in a variety of clinical situations including cardio pulmonary arrest, severe pulmonary edema, drug ingestion with severe central nervous system depression and hypo ventilation, metabolic acidosis with potassium depletion producing paralysis of the respiratory muscles (11).

In these cases, serum HCo_3^- is usually low, PCo_2 is normal or elevated and the resultant pH is usually low. The CO_2 retention prevents respiratory compensation for the metabolic acidosis and metabolic process prevents compensation for respiratory acidosis. Significant acidemia can seriously impair cardiac function and leads to cardio vascular collapse (12).

Specific therapy must be initiated aggressively to correct the acidemia by simultaneously treating the metabolic acidosis with bicarbonate and the respiratory acidosis with measures to improve ventilation in this setting, hyperkalemia is a serious problem.

2) RESPIRATORY ALKALOSIS + METABOLIC ALKALOSIS :

This mixed disorder is commonly present in patients with hepatic failure, who are placed on diuretics or nasogastric suction. It is also commonly in critically ill patients who require ventilator support and or

given diuretics or nasogastric suction. In this cases, serum HCo_3^- is usually elevated. PCo_2 is normal or low and pH is extreme alkalemia, adversely affect both cerebral and peripheral haemodynamics (13).

Here metabolic process prevents compensation for respiratory alkalosis and hyper ventilation prevents compensation for metabolic alkalosis. In order to return the pH toward normal, therapy theoretically should again be directed at alleviating both disorders simultaneously. Treatment of metabolic alkalosis with volume, chloride and potassium replacement should be initiated.

3) RESPIRATORY ALKALOSIS + METABOLIC ACIDOSIS

Clinical settings in which this combination can be found include septic shock, pulmonary embolism, and renal failure with sepsis and salicylate ingestion. The metabolic acidosis in these cases is frequently of the high anion gap variety (14).

Serum HCo_3^- is markedly diminished, PCo_2 is also low. pH may be normal or mildly deviated depending upon individual disturbances. Examining the respiratory compensation for the metabolic acidosis is critical in this situation, since the respiratory alkalosis will be missed until it is realized that ventilation is greater than predicted value. Specific treatment

aimed at correcting the pH is not necessary. In fact bicarbonate therapy is contraindicated in severe respiratory alkalosis. Therefore the main value in recognizing this disturbance is more for its diagnostic potential rather than treatment purpose.

4) RESPIRATORY ACIDOSIS + METABOLIC ALKALOSIS

It is present most commonly in patients with chronic lung disease and CO_2 retention. In these patients metabolic alkalosis arises because of vomiting or treatment with diuretics under low salt diet (15).

Serum HCo_3^- is raised, PCo_2 is also raised, pH may be normal. So it is important to recognize and treat primary metabolic alkalosis with volume, chloride and potassium replacement. Since the elevated bicarbonate may itself depress respiration (16).

pH AND SURVIVAL :

Severe acidemia is defined as pH less than 7.20 and severe alkalemia as pH more than 7.60. Adverse consequences can occur independent of whether the acidemia is of metabolic, respiratory or mixed origin (17).

MAJOR CONSEQUENCES OF SEVERE ACIDEMIA ARE :

- Cardio Vascular

  Impairment of Cardiac contractility

  Arteriolar dilatation

  Veno constriction

  Increased Pulmonary vascular resistance

  Reduction of Cardiac output

  Reduced hepatic and renal blood flow (18)

  Sensitization to reentrant arrhythmia

  Reduced threshold for ventricular fibrillation (19)

- Respiratory

  Hyperventilation

  Muscle fatigue

- Metabolic

  Insulin Resistance

  Hyperkalemia

  Increased protein degradation (20)

  Inhibition of anaerobic glycolysis (21)

- Cerebral

    Inhibition of metabolism and cell volume regulation

    Obtundation and coma

ADVERSE CONSEQUENCES OF SEVERE ALKALEMIA

- Cardio Vascular

    Arteriolar constriction (22)

    Reduction in coronary blood flow

    Reduced anginal threshold

    Predisposes to SVT/VT

- Respiratory

    Hypoventilation

    Hypercapnia and hypoxia.

- Metabolic

    Stimulation of anaerobic glycolysis

    Hypokalemia (23)

    Reduced plasma ionized calcium

- Cerebral

    Reduced blood flow

    Tetany, Seizure, Lethargy, stupor

RESPIRATORY FAILURE:

It is a condition in which respiratory system fails in one or both of its function namely, gas exchange, oxygen delivery and carbon di oxide elimination. Respiratory failure may be acute or chronic, the clinical presentation of patients with acute and chronic respiratory failure are quite different. While acute respiratory failure is characterized by life threatening derangements in arterial blood gases and acid base status, chronic failure are more indolent and clinically in apparent (24).

In the healthy adult at C level (760 mm atm. Pressure), breathing room air (FiO_2 0.21), the normal P_aO_2 is stated to be 97 mm Hg (25).Hypoxemia is defined as an arterial PO_2 of less than 80 mm Hg, hypercapnia when arterial PCO_2 more than 45 mm Hg (26)

ADULT VALUES FOR P_aO_2 AND S_aO_2

	P_aO_2	S_aO_2
Normal	97	97
Normal Range	>= 80	>= 95
Hypoxia	< 80	< 95
Mild	60 – 79	90 – 94
Moderate	40 – 59	75 – 89
Severe	< 40	< 75

Hypoxia and Hypercapnia stimulate chemoreceptors in arterial circulation (peripheral receptor) and ventro lateral medulla (central), which increases the motor activity of the respiratory skeletal muscle of chest wall and upper airways.

Under isocapnic conditions ventilation increases in curvilinear fashion as PO_2 falls (27). However hypoxic response depends on prevailing level of PCO_2 . Hypercapnia increase the hypoxic response by shifting the PO_2 threshold to higher level (28). In contrast to hypoxic response, the response to hypercapnia under isotoxic condition is on linear fashion (29).

Hypoxic and hypercapnic stimulous acts multiplicatively to enhance the motor activity of the respiratory muscle. Chemo sensitivity to hypoxia and hypercapnia are heredito-familial and vary individually (30). The chemo sensitivity response decreases with age, which explains predilection of respiratory failure in elderly.

Small changes in PO_2 (5 – 15 mm Hg) occurring gradually over days or week leaving pH at 7.25 – 7.30 are well tolerated but rapid PO_2 changes and pH less than 7.25 are life threatening and conveys the needs for ventilatory support.

ACID BASE DISORDERS IN OPC POISONING

The most common acid base disorders seen in OPC poisoning is simple metabolic alkalosis. Loss of Hcl from the upper gastro intestinal tract either from vomiting or nasogastric suction increases serum HCO_3^- and produces metabolic alkalosis that is sustained until the chloride losses are replenished. For each H^+ lost by the stomach, a new HCO_3^- is generated in the body fluids. The accompanying chloride losses sustain the increase in serum bicarbonate by altering renal transport processes and promoting renal K^+ losses (31).

These agents depress the heart rate or myocardial contractility directly and cause confusion, CNS depression, muscle weakness, salivation, lacrimation, diaphoresis, urinary incontinence and muscle fasciculation. Later reduced ventilatory drive and decreases minute volume resulting in a respiratory acidosis (32).

ACID BASE DISORDERS IN COPD

The most common acid base disorders seen in COPD is compensated respiratory acidosis (33). More severe the COPD, more likely that hypoxia and hypercapnia will be present CO_2 retention is compensated by renal retention of bicarbonate ions, normalizing pH. Progressive airway

obstruction leads to compensatory metabolic alkalosis (34). The presence of elevated bicarbonate level in a patient with hypercapnic failure indicates indulging chronic hypercapnia.

Respiratory alkalosis is the most common acid base disorders in acute asthma (35). Respiratory alkalosis causes broncho constriction, reduced cerebral blood flow and neuro excitatory symptoms. Acute respiratory acidosis can give rise to CO_2 narcosis.

ACID BASE DISORDERS IN DIABETIC KETO ACIDOSIS

During the development of ketosis in a diabetic individual, the keto acids released into the extra cellular fluid are titrated by bicarbonate ions. This buffering results in increased plasma unmeasured anions and cause high anion gap acidosis (36). In DKA each increase in anion gap from retained keto acid should be identical to decrease in plasma HCO_3^-. Thus in uncomplicated DKA, the increase in anion gap above its normal value should be equal to decrease in HCO_3^- (37). Plasma HCO_3^- must be reduced in DKA, unless it is complicated by coexisting respiratory acidosis or metabolic alkalosis. Acidemia is the rule in DKA, unless coexisting metabolic alkalosis. As the renal threshold of plasma keto acids is low, its products can reach 1000 – 2000 meq per day. Urinary loss of keto acids may

be enormous which is associated with sodium and potassium excretion which is replenished by chloride ions absorption causing net effect of hyper chloremic acidosis.

ACIDBASE DISORDERS IN SEPSIS

American college of chest physician and critical care medicine formulated a working definition of sepsis as a clinical evidence of infection with temperature of $> 38^{o}$ c or $< 36^{o}$ C, respiratory rate > 20 / mt, heart rate > 90 / mt, WBC > 12000 or < 4000 with > 10 % immature band forms (38). Severe sepsis is defined if sepsis associated with organ dysfunction like hypotension, hypoxia, oliguria, confusion, metabolic acidosis and DIC. The hallmark of sepsis is wide spread peripheral vaso dilatation due to nitric oxide production in response to cytokines, causing loss of homeostatic regulation of tissue blood flow (39). Thus much of circulating blood volume is shunted through capillary beds bypassing deep tissue and reducing the opportunity for O_2 extraction. This will in turn exacerbate tissue hypoxia and cause metabolic acidosis. Lung is the most vulnerable organ in sepsis. TNF alpha, platelet aggregating factor, IL-8 play prominent role in development of ARDS. Neutrophils degranulation causing loss of endothelial integrity and accumulation of fluid producing impaired gas

exchange and hypoxia. Typically respiratory alkalosis occurs yearly and metabolic acidosis late. The degree of acidosis is a marker of severity of illness. The onset of hypoxia indicates the severe disease and high risk for ARDS.

ACID BASE DISORDERS IN CHRONIC RENAL FAILURE

An individual ingeting a normal diet produces about 1 meq of H^+ ions / kg body weight (40). Kidney is responsible for excretion of these metabolic H^+ ions. A normal kidney excretes 60% H^+ ions as ammonium and remaining 40% as titrable acid. As GFR falls, metabolic H^+ ions balance is maintained for as long as residual nephrons are able to increase H^+ ions excretion is proportional to fall in GFR. As GFR falls less than 30 ml / min, decrease in ammonium excretion causes metabolic acidosis. Both hyperchloremic metabolic acidosis and anion gap acidosis can complicate renal failure. Hyperchloremic metabolic acidosis can occur only if positive H^+ ions balance develops, before GFR has fallen sufficiently.

Here fall in serum HCO_3^- is matched by increased in chloride ion concentration. Individual with interstitial renal disease are particularly likely to develop this form of metabolic acidosis. In more advanced renal failure, organic acids are retained and an anion gap metabolic acidosis supervenes.

MATERIALS AND METHODS

This study is descriptive study conducted in 100 patients admitted in the intensive care and toxicology units, Government General Hospital, Chennai.

The study was conducted between September 2006 – August 2007 for a period of one year.

INCLUSION CRITERIA

Acid base abnormalities is evaluated by using a five step approach.

Step 1: Validity is checked by using formula $H^+ = 24 \times PCO_2 / HCO_3^-$

Step 2: Minimum diagnosis is obtained using pH.

Step 3: To find out simple or mixed acid base disorder.

Step 4: To calculate anion gap ($AG = Na^+ - (HCO_3^- + Cl^-)$).

Step 5: To Identify triple acid base disorder.

If a primary acidosis or alkalosis is present, the expected degree of compensation can be predicted using following equations.

SIMPLE ACID BASE DISORDER :

Metabolic Acidosis :

Expected PCO_2 = 1.5 x (HCO_3^- + 8 $\pm$ 2)

Metabolic Alkalosis :

Expected PCO_2 = 0.9 x (HCO_3^- + 16 $\pm$ 2)

If measured PCO_2 is less than expected PCO_2 then respiratory alkalosis is present. If measured PCO_2 is greater than expected PCO_2 then respiratory acidosis is present.

Respiratory Acidosis :

Plasma HCO_3 will increase by 1 meq / L for each 10 mm Hg increase PCO_2 in acute cases and 4 meq / L in chronic cases.

Respiratory Alkalosis :

Plasma HCO_3 will increase by 2 meq / L for each 10 mm Hg decrease PCO_2 in acute cases and 4 meq / L in chronic cases.

MIXED ACID BASE DISORDERS

Lack of appropriate compensation for a single acid base disturbance suggests mixed acid base disorder.

EXCLUSION CRITERIA

1. All Surgical patients
2. All obstetrics and Gynaecological patients

METHODS:

OBTAINING A ARTERIAL BLOOD SAMPLE :

Based on safety, accessibility and patients comfort, site for obtaining arterial blood samples is chosen. Radial, femoral and brachial arteries are the ones from which blood samples most commonly taken. The radial artery is used most often, because it is superficially located and well supported by collateral circulation by ulnar artery. If radial artery is inaccessible, brachial or femoral artery is punctured.

The syringe is adequately heparinised to prevent the sample clotting. About 0.25 ml of heparin is drawn up in to the syringe. The plunger is withdrawn to allow the heparin to coat the wall of the syringe and then the heparin is completely expelled.

EQUIPMENTS REQUIRED :

1. Skin preparation fluid – alcohol or iodine
2. Syringe of size 21 G – 2 ml containing 0.5 % plain lignocaine.
3. A needle size 23 G is attached to the heparinised syringe.
4. A cap to seal the syringe.
5. Ice packs if transferred to the lab take more than 5 minutes.

PROCEDURE :

After explaining to the patient and obtaining their consent, pulse in the desired area is identified. At the maximum point of pulsation the lignocaine is infiltrated subcutaneously.

Using 23 G needle the heparinised syringe is inserted at an angle of 20 to 30 degrees towards radial artery. About 2 ml of blood is aspirated and the syringe is sealed with cap without any air bubbles. Tight pressure is applied over the punctured side for two minutes.

SOURCES OF ERROR:

1. Calibration error of the machine.
2. Any sample with more than minor air bubble should be discarded, as they will significantly lower the PCO_2, with increase in pH and PO_2 .
3. Too much heparin will alter pH, PCO_2 and PO_2 .
4. Delay in analysis causes increase in PCO_2.
5. If there is delay and sample is not adequately cooled will give rise to erroneous results.

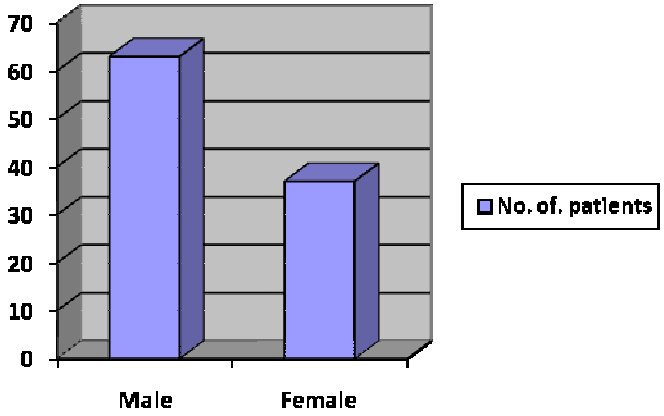
Along the arterial blood gas, serum electrolytes, blood sugar, blood urea, serum creatinine were also sent.

OBSERVATIONS AND RESULTS

Table – 1

CATEGORY OF CASES INCLUDED FOR THE STUDY & PREVALENCE OF ACIDBASE DISORDERS

Sex Distribution in the study group



100 patients were taken up to study
63 patients were male
37 patients were female
Mean age of all patients was 39.89

Sl no	SUB CATEGORY OF CASES	NUMBER	PERCENTAGE
1	Toxicology :		
	Organo phosphorus compound poisoning	17	17%
	Alcoholic intoxication	6	6%
2	Infection :		
	Sepsis	13	13%
3	Diseases of metabolic derangements		
	Diabetic Keto acidosis	10	10%
	Chromic Kidney disease	10	10%
	Chromic obstractive pulmonary disease	9	9%
	Decompensated Liver disease	4	4%
4	BITES AND STINGS		
	Scorpion sting	6	6%
	Snake bite	5	5%
5	Miscellaneous	20	20%
	Total Cases	100	

Table 2

ACID BASE DISORDER IDENTIFIED

Total	***100***
Simple	40
Mixed	60

Mixed acid base disorder accounts for 60%

Table 3

SIMPLE ACID BASE DISORDER

TOTAL	***40***
Metabolic acidosis	19
Metabolic alkalosis	14
Respiratory alkalosis	4
Respiratory acidosis	3

Metabolic acidosis is the most common simple acid base disorder.

Table 4

MIXED ACID BASE DISORDER

Metabolic alkalosis + Respiratory alkalosis	***22***
Metabolic acidosis + Respiratory acidosis	19
Metabolic alkalosis + Respiratory acidosis	9
Metabolic acidosis + Respiratory alkalosis	6
Metabolic acidosis + Met.alkalosis + Resp.alkalosis	3
Met.acidosis + Met.alkalosis + Resp.acidosis	1
Total	60

The most common mixed acid base disorder is combination of

Metabolic alkalosis + Respiratory alkalosis

Table 5

Acid base disorder	Opc poisoning	Sepsis	DKA	CKD	COPD	Alcholic intoxication	Scorpion Sting	Snake Bite	DCLD	Miscellanious	Total
Simple Met. Acidosis	3	4	4	3			2	1		2	19
Simple Met. Alkalosis	6	3								5	14
Simple resp. Acidosis					2					1	3
Simple resp. Alkalosis		1				2				1	4
M. alkalosis + R.alkalosis	3	5		2		2		2	3	5	22
M. acidosis + R. acidosis	3		1	1	4		4	2		4	19
M.alkalosis + R. acidosis	2				3	1			1	2	9
M. acidosis + R. alkalosis			2	3		1					6
M. acd + M.alk + resp.alkalaosis			3								3
M. acd + M.alk + resp.acidosis				1							1
Total	17	13	10	10	9	6	6	5	4	20	100
Expired	3	8	-	7	4	-	2	-	2	6	32

Organo phosphorus compound poisoning is the most common acid base disturbance in our study followed sepsis , DKA and CKD.

Table 6

PREVALENCE OF INDIVIDVAL ACID BASE DISTURBANCES :

METABOLIC ACIDOSIS

Simple metabolic acidosis	***Mixed Metabolic Acidosis***			***Total***
19	+ Res. Alkalosis	+ Res. Alkalosis	+ Met. alkalosis	48
	19	6	4	

48% of patients had metabolic acidosis either in simple or mixed form

Table 7

RESPIRATORY ALKALOSIS

Simple Respiratory Alkalosis	Mixed Respiratory Alkalosis			Total
4	+ met. acidosis	+ met. alkalosis	+ met. acidosis + met. alkalosis	35
	6	22	3	

35% of patients had respiratory alkalosis either in simple or mixed form.

Table 8

METBOLIC ALKALOSIS

Simple Metabolic Alkalosis	Mixed Metabolic Alkalosis			Total
14	+ Res. acidosis	+Res. alkalosis	+Met. Acidosis	49
	9	22	4	

49% of patients had metabolic either in simple or mixed form

Table 9

RESPIRATORY ACIDOSIS

Simple respiratory acidosis	Mixed respiratory acidosis			Total
3	+ Met . alkalosis	+ Met . acidosis	+ Met. Alkalosis + Met. Acidosis	32
	9	19	1	

Respiratory acidosis is the least common disturbance accounting for 32 %

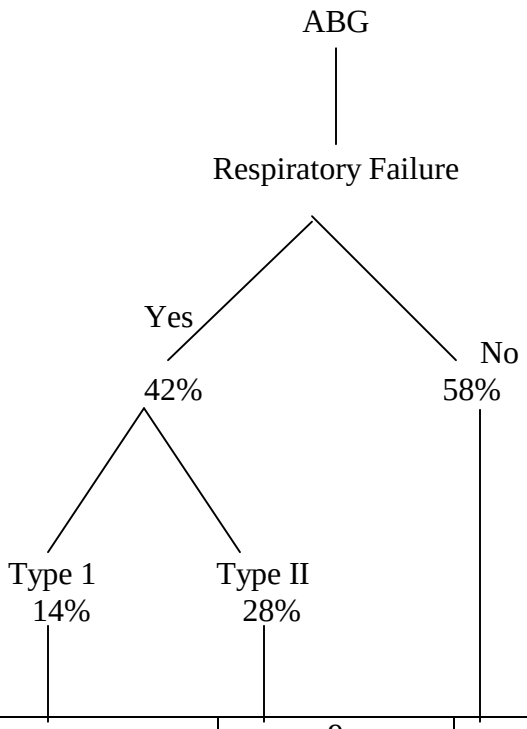
Table 10

pH AND SURVIVAL:

pH	< 7.2	7.2 - 7. 6			> 7.6	Total
		7.2 – 7.35	7.35 – 7.45	7.45 – 7.6		
Total	13	27	22	37	1	100
Expired	10	7	3	11	1	32
%	76	25	13	29	100	32

The mortality rate is higher in extreme acidemia and alkalemia

Table 11
ACID BASE DISORDER AND RESPIRATORY FAILURE



				Total Cases
COPD	-	9	-	9
Sepsis	4	2	7	13
OPC Poisoning	2	7	8	17
CKD	1	-	9	10
DKA	-	-	10	10
Snake Bite	1	2	2	5
Scorpion Sting	1	2	3	6
Alcoholic Intoxication	-	1	5	6
DCLD	1	1	2	4
Miscellaneous	4	4	12	20
	14	28	58	100
Simple	9	8	23	40
Mixed	5	20	35	60

Table 12

ACID BASE DISORDER AND COPD

TOTAL		9
Expired		4
Respiratory Failure		9
	Type 1	-
	Type 2	9
PH	< 7 . 2	2
	7.2 - 7.6	7
	> 7 . 6	0

Simple		2
	Met . acidosis	0
	Met . alkalosis	0
	Res . acidosis	2
	Res . alkalosis	0
Mixed		7
	Met . alkalosis + Res. Alkalosis	0
	Met . acidosis + Res. acidosis	4
	Met. alkalosis + Res . acidosis	3
	Met.acidosis + Res. Alkalosis	0
	Met.acid + M.alk + Res.alkalosis	0
	M.acd + M.alk+Res.acidosis	0

All patients with COPD had Respiratory Failure (Type 2)

Table 13

ACID BASE DISORDERS IN COPD

No	Pt	Acid Base disturbance	PH	Resp . failure	Survial
1	1	Simple Resp. acidosis	7.42	Type II	-
2	26	Mixed Resp+ Met. acidosis	7.22	Type II	-
3	27	Mixed Resp+ Met. acidosis	7.18	Type II	Expired
4	52	Mixed Resp+ Met. acidosis	7.05	Type II	Expired
5	53	Mixed Resp acidosis + M.alkalosis	7.44	Type II	-
6	67	Mixed Resp acid + Met.alkalosis	7.34	Type II	-
7	77	Mixed Resp acid + Met.alkalosis	7.51	Type II	Expired
8	78	Mixed Res+ Met acidosis	7.20	Type II	Expired
9	89	Simple Resp.acidosis	7.31	Type II	-

Out of 9 patients, 4 had expired accounting for a mortality of 44%

Table – 14

SEPSIS AND ACID BASE DISTURBANCE

TOTAL		13
Expired		8
Respiratory Failure		6
	Type 1	4
	Type 2	2
PH	< 7 . 2	0
	7.2 - 7.6	12
	> 7 . 6	1

Simple		8
	Met . acidosis	4
	Met . alkalosis	3
	Res . acidosis	0
	Res . alkalosis	1
Mixed		5
	Met . alkalosis + Res. Alkalosis	5
	Met . acidosis + Res. Acidosis	0
	Met. alkalosis + Res . acidosis	0
	Met.acidosis + Res. Alkalosis	0
	Met.acid + M.alkalosis + Res.alkalosis	0
	M.acid + M.alkalosis+Res.acidosis	0

6 patients had Respiratory failure (Type 1 – 4 , Type 2 -2)

Table – 15

ACID BASE DISORDERS IN SEPSIS

S.No	Pt	Acid Base Disorder	pH	Respiratory Failure	Survival
1	2	Simple Respiratory Alkalosis	7.54	Type 1	Expired
2	28	Mixed Met + Resp Alkalosis	7.58	-	Expired
3	29	Simple Met. acidosis	7.31	-	
4	54	Simple met . acidosis	7.32	Type 1	
5	68	Mixed met + Res Alkalosis	7.48	-	
6	69	Simple metabolic alkalosis	7.43	Type 2	
7	79	Mixed met + Resp alkalosis	7.62	Type 1	Expired
8	80	Mixed met + Res alkalosis	7.52	-	Expired
9	81	Simple met. alkalosis	7.46	Type 2	Expired
10	90	Simple met. acidosis	7.32	-	Expired
11	91	Mixed met. + resp. alkalosis	7.51	Type 1	
12	93	Simple met alkalosis	7.54	-	Expired
13	97	Simple met acidosis	7.38	-	Expired

8 Expired out of 13 patients with mortality rate of 61%.

Table – 16

ACID BASE DISORDERS IN SEPSIS

S.No	Pt	Acid Base Disorder	pH	Respiratory Failure	Survival
1	2	Simple Respiratory Alkalosis	7.54	Type 1	Expired
4	54	Simple met . acidosis	7.32	Type 1	
6	69	Simple metabolic alkalosis	7.43	Type 2	
7	79	Mixed met + Resp alkalosis	7.62	Type 1	Expired
9	81	Simple met. alkalosis	7.46	Type 2	Expired
11	91	Mixed met. + resp. alkalosis	7.51	Type 1	

Table – 17

ACID BASE DISORDERS IN SEPSIS

S.No	Pt	Acid Base Disorder	pH	Respiratory Failure	Survival
2	28	Mixed Met + Resp Alkalosis	7.58	-	Expired
3	29	Simple Met. acidosis	7.31	-	
5	68	Mixed met + Res Alkalosis	7.48	-	
8	80	Mixed met + Res alkalosis	7.52	-	Expired
10	90	Simple met. acidosis	7.32	-	Expired
12	93	Simple met alkalosis	7.54	-	Expired
13	97	Simple met acidosis	7.38	-	Expired

Table – 18

ACID BASE DISTURBANCE IN ORGANO PHOSPHORUS COMPOUND POISONING

TOTAL		17
Expired		3
Respiratory Failure		9
	Type 1	2
	Type 2	7
PH	< 7 . 2	3
	7.2 - 7.6	14
	> 7 . 6	0

Simple		9
	Met . acidosis	3
	Met . alkalosis	6
	Res . acidosis	0
	Res . alkalosis	0
Mixed		8
	Met . alkalosis + Res. Alkalosis	3
	Met . acidosis + Res. Acidosis	3
	Met. alkalosis + Res . acidosis	2
	Met.acidosis + Res. Alkalosis	0
	Met.acid + M.alkalosis + Res.alkalosis	0
	M.acid + M.alkalosis+Res.acidosis	0

Simple metabolic alkalosis is most common in OPC poisoning accounting for 35% of cases

Table 19

ACID BASE DISORDERS IN OPC POISONING

No	Pt	Acid Base disturbance	PH	Resp . failure	Survival
1	15	Mixed Res acid + Met alkalosis	7.33	Type II	-
2	16	Simple Metabolic alkalosis	7.47	Type II	-
3	17	Mixed Met alkalosis+Resp acidosis	7.38	Type II	-
4	38	Mixed Met + Res acidosis	6.70	Type II	Expired
5	39	Simple metabolic alkalosis	7.48	Type II	-
6	40	Mixed Met + Resp. alkalosis	7.49	-	-
7	41	Mixed Met + Resp.alkalosis	7.58	-	-
8	57	Simple Met alkalosis	7.54	Type II	-
9	71	Simple Met acidosis	7.01	Type I	Expired
10	72	Simple Met alkalosis	7.46	-	-
11	73	Mixed Met + Resp.alkalosis	7.53	-	-
12	84	Simple Met alkalosis	7.49	-	-
13	85	Simple Met alkalosis	7.46	-	-
14	95	Simple Met acidosis	7.30	Type I	-
15	96	Mixed Met + Res acidosis	7.09	-	-
16	99	Mixed Met + Res acidosis	6.93	Type II	Expired
17	100	Simple Met acidosis	7.34	-	-

3 out of 17 patients expired with mortality rate of 17%

Table 20

ACID BASE DISORDERS IN OPC POISONING

No	Pt	Acid Base disturbance	PH	Resp . failure	Survival
1	15	Mixed Res acid + Met alkalosis	7.33	Type II	-
2	16	Simple Metabolic alkalosis	7.47	Type II	-
3	17	Mixed Met alkalosis+Resp acidosis	7.38	Type II	-
4	38	Mixed Met + Res acidosis	6.70	Type II	Expired
5	39	Simple metabolic alkalosis	7.48	Type II	-
8	57	Simple Met alkalosis	7.54	Type II	-
9	71	Simple Met acidosis	7.01	Type I	Expired
14	95	Simple Met acidosis	7.30	Type I	-
16	99	Mixed Met + Res acidosis	6.93	Type II	Expired

Table 21

ACID BASE DISORDERS IN OPC POISONING

No	Pt	Acid Base disturbance	PH	Resp . failure	Survival
6	40	Mixed Met + Resp. alkalosis	7.49	-	-
7	41	Mixed Met + Resp.alkalosis	7.58	-	-
10	72	Simple Met alkalosis	7.46	-	-
11	73	Mixed Met + Resp.alkalosis	7.53	-	-
12	84	Simple Met alkalosis	7.49	-	-
13	85	Simple Met alkalosis	7.46	-	-
15	96	Mixed Met + Res acidosis	7.09	-	-
17	100	Simple Met acidosis	7.34	-	-

Table – 22
ACID BASE DISTURBANCE AND CKD

TOTAL		10
Expired		7
Respiratory Failure		1
	Type 1	1
	Type 2	0
PH	< 7 . 2	2
	7.2 - 7.6	8
	> 7 . 6	0

Simple		3
	Met . acidosis	3
	Met . alkalosis	0
	Res . acidosis	0
	Res . alkalosis	0
Mixed		7
	Met . alkalosis + Res. Alkalosis	2
	Met . acidosis + Res. Acidosis	1
	Met. alkalosis + Res . acidosis	0
	Met.acidosis + Res. Alkalosis	3
	Met.acid + M.alkalosis + Res.alkalosis	0
	M.acid + M.alkalosis+Res.acidosis	1

Metabolic acidosis is the most common acid base disturbance in CKD

Table – 23

ACID BASE DISORDERS IN CKD

S.No	Pt	Acid Base Disorder	pH	Respiratory Failure	Survival
1	3	Mixed Met.Acd + Res.Alk	7.39	-	-
2	30	Mixed Met.Acd + Res.Alk	7.52	-	-
3	31	Simp Met.Acd	7.23	-	-
4	55	Mixed Met.Acd + Res.Alk	7.5	-	Expired
5	70	Mixed Met.Alk + Res.Alk	7.58	-	Expired
6	82	Mixed Met.Acd + Res.Alk	7.08	-	Expired
7	83	Triple Met.Acd + Met.Alk + Res.Acd	7.3	-	Expired
8	92	Simp Met.Acd	7.32	TYPE I	Expired
9	94	Mixed Met.Alk + Res.Alk	7.44	-	-
10	98	Simple met. acidosis	7.1	-	Expired

7 out of 10 patients expired with mortality rate of 70%.

Table – 24

ACID BASE DISTURBANCES AND DKA

TOTAL		10
Expired		0
Respiratory Failure		0
	Type 1	0
	Type 2	0
PH	< 7 . 2	0
	7.2 - 7.6	10
	> 7 . 6	0

Simple		4
	Met . acidosis	4
	Met . alkalosis	0
	Res . acidosis	0
	Res . alkalosis	0
Mixed		6
	Met . alkalosis + Res. Alkalosis	0
	Met . acidosis + Res. Acidosis	1
	Met. alkalosis + Res . acidosis	0
	Met.acidosis + Res. Alkalosis	2
	Met.acid + M.alkalosis + Res.alkalosis	3
	M.acid + M.alkalosis+Res.acidosis	0

Mixed acid base disorder is the most common disturbance in DKA accounting for 60% of cases.

Table – 25

ACID BASE DISORDERS IN DKA

S.No	Pt	Acid Base Disorder	pH	Respiratory Failure	Survival
1	12	Simp Met.Acd	7.30	-	-
2	13	Simp Met.Acd	7.27	-	-
3	36	Simp Met.Acd	7.31	-	-
4	37	Simp Met.Acd	7.3	-	-
5	50	Mixed Met.Acd + Res.Acd	7.23	-	-
6	51	Mixed Met.Alk + Res.Alk	7.38	-	-
7	65	Mixed Met.Acd + Met.Alk + Res.Alk	7.42	-	-
8	66	Mixed Met.Acd + Met.Alk + Res.Alk	7.42	-	-
9	76	Mixed Met.Acd + Res.Alk	7.43	-	-
10	88	Mixed Met.Acd + Res.Alk	7.4	-	-

All patients survived without any mortality

Table – 26

ACID BASE DISTURBANCES AND SCORPION STING

TOTAL		6
Expired		2
Respiratory Failure		3
	Type 1	1
	Type 2	2
PH	< 7 . 2	3
	7.2 - 7.6	3
	> 7 . 6	0

Simple		2
	Met . acidosis	2
	Met . alkalosis	0
	Res . acidosis	0
	Res . alkalosis	0
Mixed		4
	Met . alkalosis + Res. Alkalosis	0
	Met . acidosis + Res. Acidosis	4
	Met. alkalosis + Res . acidosis	0
	Met.acidosis + Res. Alkalosis	0
	Met.acid + M.alkalosis + Res.alkalosis	0
	M.acid + M.alkalosis+Res.acidosis	0

3 out of 6 patients had respiratory failure in scorpion sting. The combination of metabolic acidosis and respiratory acidosis is the most common disturbance in scorpion sting

Table – 27

ACID BASE DISTURBANCES AND SNAKE BITE

TOTAL		5
Expired		0
Respiratory Failure		3
	Type 1	1
	Type 2	2
PH	< 7 . 2	1
	7.2 - 7.6	4
	> 7 . 6	0

Simple		1
	Met . acidosis	1
	Met . alkalosis	0
	Res . acidosis	0
	Res . alkalosis	0
Mixed		4
	Met . alkalosis + Res. Alkalosis	2
	Met . acidosis + Res. Acidosis	2
	Met. alkalosis + Res . acidosis	0
	Met.acidosis + Res. Alkalosis	0
	Met.acid + M.alkalosis + Res.alkalosis	0
	M.acid + M.alkalosis+Res.acidosis	0

3 out of 5 patients had respiratory failure in snake bite. Type II failure is the most common respiratory failure in snake bite accounting for 66% of cases.

Table – 28

ACID BASE DISTURBANCES AND ALCOHOLIC INTOXICATION

TOTAL		6
Expired		0
Respiratory Failure		1
	Type 1	0
	Type 2	1
PH	< 7 . 2	0
	7.2 - 7.6	6
	> 7 . 6	0

Simple		2
	Met . acidosis	0
	Met . alkalosis	0
	Res . acidosis	0
	Res . alkalosis	2
Mixed		4
	Met . alkalosis + Res. Alkalosis	2
	Met . acidosis + Res. Acidosis	0
	Met. alkalosis + Res . acidosis	1
	Met.acidosis + Res. Alkalosis	1
	Met.acid + M.alkalosis + Res.alkalosis	0
	M.acid + M.alkalosis+Res.acidosis	0

Respiratory alkalosis is the most common disturbance in alcoholic intoxication.

Table – 29

ACID BASE DISORDERS IN SCORPION STING

S.No	Pt	Acid Base Disorder	pH	Respiratory Failure	Survival
1	21	Mixed Met.Acd + Res.Acd	6.94	-	Expired
2	46	Simp Met.Acd	7.22	-	-
3	61	Simp Met.Acd	7.4	Type I	-
4	74	Met.Acd + Res.Acd	6.95	Type II	Expired
5	86	Met.Acd + Res.Acd	7.17	Type II	-
6	87	Met.Acd + Res.Acd	7.23	-	-

Table – 30

ACID BASE DISORDERS IN SNAKE BITE

S.No	Pt	Acid Base Disorder	pH	Respiratory Failure	Survival
1	20	Mixed Met.Acd + Res.Acd	7.20	Type II	-
2	44	Met.Alk + Res.Alk	7.50	-	-
3	45	Simp Met.Acd	7.41	Type I	-
4	60	Met.Acd + Res.Acd	7.17	Type II	-
5	75	Met.Alk + Res.Alk	7.59	-	-

Table – 31

ACID BASE DISORDERS IN ALCOHOLIC INTOXICATION

S.No	Pt	Acid Base Disorder	pH	Respiratory Failure	Survival
1	18	Simp Res.Alk	7.50	-	-
2	19	Simp Res.Alk	7.50	-	-
3	42	Mixed Met.Alk + Res.Alk	7.54	-	-
4	43	Mixed Met.Alk + Res.Alk	7.54	-	-
5	58	Mixed Met.Acd + Res.Alk	7.24	-	-
6	59	Mixed Met.Alk + Res.Alk	7.44	Type II	-

Table – 32

ACID BASE DISTURBANCES AND DCLD

TOTAL		4
Expired		2
Respiratory Failure		2
	Type 1	1
	Type 2	1
PH	< 7 . 2	0
	7.2 - 7.6	4
	> 7 . 6	0

Simple		0
	Met . acidosis	0
	Met . alkalosis	0
	Res . acidosis	0
	Res . alkalosis	0
Mixed		4
	Met . alkalosis + Res. Alkalosis	3
	Met . acidosis + Res. Acidosis	0
	Met. alkalosis + Res . acidosis	1
	Met.acidosis + Res. Alkalosis	0
	Met.acid + M.alkalosis + Res.alkalosis	0
	M.acid + M.alkalosis+Res.acidosis	0

Combination of metabolic alkalosis and respiratory alkalosis is the most common disturbance in DCLD

Table– 33

ACID BASE DISTURBANCES IN MISCELLANEOUS GROUP

TOTAL		20
Expired		6
Respiratory Failure		8
	Type 1	4
	Type 2	4
PH	< 7 . 2	1
	7.2 - 7.6	19
	> 7 . 6	0

Simple		8
	Met . acidosis	2
	Met . alkalosis	4
	Res . acidosis	1
	Res . alkalosis	1
Mixed		12
	Met . alkalosis + Res. Alkalosis	5
	Met . acidosis + Res. Acidosis	4
	Met. alkalosis + Res . acidosis	2
	Met.acidosis + Res. Alkalosis	0
	Met.acid + M.alkalosis + Res.alkalosis	0
	M.acid + M.alkalosis+Res.acidosis	0

Combination of metabolic alkalosis and respiratory alkalosis is the most common disturbance in this group

Table – 34
ACID BASE DISORDERS IN DCLD

S.No	Pt	Acid Base Disorder	pH	Respiratory Failure	Survival
1	4	Mixed Met.Alk + Res.Alk	7.52	-	Expired
2	32	Met.Alk + Res.Acd	7.41	Type II	-
3	33	Met.Alk + Res.Alk	7.54	-	Expired
4	56	Met.Alk + Res.Alk	7.54	Type I	-

Out of 4 patients 2 patients had expired

Table – 35

ACID BASE DISORDERS IN MISCELLANEOUS GROUP

S.No	Pt	Diagnosis	Acid Base Disorder	pH	Respiratory Failure	Survival
1	5	CVA	Met.Alk + Res.Alk	7.54	Type I	-
2	6	CVA	Met.Alk + Res.Alk	7.44	-	Expired
3	34	CVA	Met.Alk + Res.Alk	7.54	Type I	-
4	7	Myasthenic Crisis	Met.Alk + Res.Acd	7.24	-	-
5	8	Antiphospholipid antibody syndrome	Met.Alk + Res.Alk	7.50	-	-
6	9	Pneumothorax	Met.Acd + Res.Acd	7.26	-	Expired
7	35	Pneumothorax	Simp Met.Alk	7.50	-	Expired
8	10	Cardiac failure	Met.Alk + Res.Alk	7.40	Type II	Expired
9	11	Pneumonia	Simp Res.Alk	7.56	-	-
10	14	Hypokalemic paralysis	Met.Alk + Res.Acd	7.38	Type II	-
11	22	Corrosive poisoning	Simp Met.Alk	7.49	Type I	-
12	47	Corrosive poisoning	Simp Res.Acd	7.29	Type II	-
13	48	Corrosive poisoning	Simp Met.Acd	7.33	=	Expired
14	62	Corrosive poisoning	Simp Met.Alk	7.49	Type I	-
15	23	Oduvanthalai poisoning	Met.Acd + Res.Acd	7.00	Type II	Expired
16	24	Attempted hanging	Met.Acd + Res.Acd	7.28	-	-
17	49	Attempted hanging	Simp Met.Alk	7.49	-	-
18	63	Attempted hanging	Met.Alk + Res.Alk	7.58	-	-
19	64	Attempted hanging	Simp Met.Acd	7.38	-	-
20	25	Sedative poisoning	Met.Acd + Res.Alk	7.43	-	-

6 out of 20 patients expired with mortality rate of 30%

Discussion

DISCUSSION

Disturbances of the acid base equilibrium occur in a wide variety of critical illnesses and are among the most commonly encountered disorders in the ICU. In addition to reflecting the seriousness of the underlying disease, these disorders have their own morbidity and mortality. This study has been undertaken to focus on acid base disturbance in critically ill patient, admitted in Government General Hospital, Chennai.

ORGANOPHOSPHOROUS COMPOUND POISONING, SEPSIS, DIABETIC KETOACIDOSIS, CHRONIC KIDNEY DISEASE AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE constitute 59% of acid base disorder in patients admitted in IMCU and toxicology units.

OPC poisoning is the most common cause of acid base disturbance in our study, which leads the tally, with 17 %. Sepsis accounts for 13%. DKA and CKD accounts for 10% each. COPD accounts for 9%. Alcoholic intoxication and scorpion sting account for 6% each. Snake bite accounts for 6%. DCLD accounts for 5 %. Corrosive poisoning and attempted hanging account for 4% each.

CVA accounts for 3%. Pneumothorax and cardiac failure account for 2% and 1% respectively.

Respiratory failure present in 42% of cases. Of which 14% are type I and 28% are type II.

The most common acid base disorder observed in our study is of mixed variety, with 60% of patients and only 40% of the patients had simple acid base disorder. This observation is consistent with the study of **Anderson et al (**1987) (41). His prospective study of over thousand consecutive ABG samples obtained from patients in intensive medical unit showed 51% of patients with mixed acid base disorder. Among the simple acid base disorder the most common is metabolic acidosis (19%). The next common disorder is metabolic alkalosis (14%) and respiratory alkalosis (4%). The least common disorder is respiratory acidosis is (3%). Our findings can be matched with studies by Madias N E et al (1982) (42). According to the study of 13,000 ABG samples, simple respiratory is the least common, which accounts for only 3%.

Among the mixed acid base disorder, the combination of metabolic alkalosis and respiratory alkalosis is the most common, which is seen in 22% of patients. Closely followed by the combination

of metabolic acidosis and respiratory acidosis which constitutes 19%. 9% of the patients had metabolic alkalosis and respiratory acidosis, while 6% had metabolic acidosis and respiratory alkalosis. Triple acid base disorder is seen in 4% of patients.

In OPC poisoning, 47% had mixed acid base disorder and 52% had simple disorder. The common acid base disorder seen in OPC poisoning is simple metabolic alkalosis. The next common disorders are simple metabolic acidosis and mixed metabolic and respiratory alkalosis and mixed metabolic and respiratory acidosis. The least common disorder is mixed metabolic alkalosis and respiratory acidosis. In all 64% of the patients had metabolic alkalosis either simple or mixed. Metabolic alkalosis occurs early and mixed metabolic and respiratory acidosis late. The degree of acidosis is a marker of severity of illness. 3 patients who had pH less than 7.2 expired, thus providing acidosis is a marker of severity of illness and prognosis. The mortality late was 17%. This correlates with the study done by **Dhadke V N** (45). According to the study of 50 patients of OPC poisoning the mortality rate was 16%. (p less than 0.001).

Respiratory failure is seen in 9 out of 17 cases. Out of which 2 had type I failure and 7 had type II failure. 3 cases had pH less than

7.2 ; 14 cases had pH between 7.2 – 7.6. This shows respiratory paralysis is a poor prognostic parameter. **Bana A K, Bhaskar Y** (46) found i) type II respiratory is the most common type of failure occurring in OPC poisoning and treatment of choice is ventilatory support. ii) ventilatory support cannot be guided by clinical assessment of respiratory inadequacy but gained only by ABG analysis. iii) Irrespective of level of consciousness, with adequate respiratory support if ABG analysis shows respiratory acidosis these patients nee ventilatory support. In OPC poisoning patients with respiratory failure showed increased mortality than those without respiratory failure (p less than 0.001). In these patients the respiratory failure is exacerbated by the development of pulmonary edema and by the retention of large amounts of respiratory secretions. A clear airway, effective removal of respiratory secretions and correction of hypoxia are essential using endotracheal intubation and assisted ventilation if necessary (47).

In sepsis 61% had simple acid base disorder and 38% had mixed acid base disorder. The most common acid base disorder is mixed metabolic and respiratory alkalosis, closely followed by the simple metabolic acidosis. Simple metabolic alkalosis and simple

respiratory alkalosis account for 3 and 1 case respectively. Out of 13 cases 8 had expired, accounting for a mortality rate of 61%. Respiratory failure is seen 6 out 13 cases. Out of which 4 cases had type I failure and 2 had type II failure. 1patient had pH more than 7.6 and the rest in between 7.2 – 7.6. In all, 46% patients had respiratory alkalosis either simple or mixed. Respiratory alkalosis occurs early and metabolic acidosis late. The degree of acidosis is a marker of severity of illness and prognosis.

The onset of hypoxia indicates severe disease and high risk for ARDS. This has been proved in our study which shows major acid base disturbance in sepsis is respiratory alkalosis; 4 patients who had type I respiratory failure expired thus providing hypoxia is a marker of severity of illness. Statistical analysis of survival in response to pH, the mean pH in survived patients is 7.42; The mean pH in expired patients is 7.54. The alkalemic pH is associated with high mortality. Several studies have shown that alkalemia is associated with high mortality in medical ward patients. Studies by **Anderson L E, Henrich W L** (41) have shown the death rate is higher among the medical patients with alkalemia and mixed metabolic and respiratory alkalosis appears to be associated with a particularly poor prognosis.

Blood gas analysis may often reveal hypoxaemia due to intra pulmonary shunting before the classical radiological appearance of ARDS develops.it is evident that patient donot usually die of hypoxaemia but from the complex disturbances that result from multiple organ system failure. Thus the aim in the management of ARDS is to support all body systems until the integrity of the alveolar capillary membrane is restored. Early recognition with appropriate pharmacological and supportive therapy favorably influence the prognosis (48). Patients treated with antimicrobials to which the organisms are sensitive do survive better than those in whom the treatment was not appropriate. However the general condition and the presence or absence of shock are powerful independent variables.

DKA is seen in 10 patients, none had respiratory failure in our study. Mixed acid base disorder is the most common abnormality seen in 60% of the cases. All of them had pH in between 7.2 – 7.6. All the patients with DKA had an element of metabolic acidosis either simple or mixed form. The most common triple acid base disorder in DKA is combination of metabolic acidosis, metabolic alkalosis and respiratory alkalosis. The underlying ketosis causes high anion gap acidosis. Vomiting or nasogastric suction can cause metabolic alkalosis in

DKA. Underlying sepsis precipitating DKA can cause respiratory alkalosis apart from neurogenic hyper ventilation.

CKD is seen in 10 patients; of which 7 patients had expired ; 1 patient had type I respiratoryfailure. The simple metabolic acidosis is seen in 3 cases and mixed acid base disorder in 7 cases.

COPD is seen in 9 patients. All of the had type II respiratory failure. Out of which 4 had expired and 7 had mixed acid base disorders (77%). The most common acid base disorder in COPD is mixed metabolic acidosis and respiratory acidosis. The next is metabolic alkalosis and respiratory acidosis. Only 2 patients had pH less than 7.2 in all respiratory acidosis seen in 100% of the cases, either simple or mixed. Respiratory in COPD is due to respiratory failure. Non-invasive ventilation is an important recent advance in the management of patients with acute respiratory failure complicating COPD. A number of large , well conducted, randomized studies have shown that non-invasive ventilation improves survival in COPD patients with an acute respiratory acidosis and reduces the need for intubation (50). The achievement of adequate alveolar ventilation, as indicated by falling $PaCo_2$, improving pH and satisfactory inspiratory chest wall movement, is the target. Patients who are likely to improve

with non-invasive ventilation will normally show both clinical and biochemical improvement within the first few hours of treatment. A falling respiratory rate, accompanied by improvement in $PaCo_2$ and pH are associated with a good outcome (51).

Alcoholic intoxication is seen in 6 patients. 66% of patients had mixed disorder of metabolic alkalosis and respiratory alkalosis. Respiratory alkalosis is seen in 2 patients. Respiratory alkalosis either simple or mixed accounts for 83% of cases. This observation is consistent with **Dobes M** (1993) (43) who did a prospective study of 77 alcoholics with delirium tremens and in 62 patients (80.5%) respiratory alkalosis was detected. Only 1 patient had type II failure . The main cause of respiratory alkalosis is rebound phenomenon of the respiratory center which causes hyper ventilation. whatever the acid base disturbance in alcoholic ketosis, administration of glucose, thiamine and rehydration is usually adequate to deal with the metabolic disturbance (52).

In scorpion sting, all six patients had an element of metabolic acidosis either simple or mixed. 3 out of 6 patients had respiratory failure (type I -1 , type II – 2). Out of the 6 cases 2 patients expired

accounting for a mortality rate of 33%. 3 of them had pH less than 7.2 and 3 had pH between 7.2 – 7.6.

In DCLD 3 out of 4 patients had an element of respiratory alkalosis. The most common acid base disorder was mixed metabolic and respiratory alkalosis. 2 patients had respiratory failure. The mortality rate was 50%.

In the miscellaneous group out of 20 patients 3 had CVA ; 4 had corrosive poisoning ; 4 cases were attempted hanging ; 1 had myasthenic crisis; 2 had pneumothorax ; 1 had pneumonia ; 1 had antiphospholipid antibody syndrome; 1 had cardiac failure ; 1 had hypokalemic paralysis ; 1 had oduvanthalai poisoning and 1 was sedative tablet poisoning.

The most common acid base disorder observed in this group is metabolic alkalosis and respiratory alkalosis. 8 patients had respiratory failure (type I – 4, type II – 4). 1 patient had pH less than 7.2 ; 19 patients had pH between 7.2 – 7.6. Out of 20 patients 6 patients had expired accounting for a mortality rate of 30%.

CKD is the most common cause of metabolic acidosis as well as mixed metabolic acidosis and respiratory alkalosis. COPD is the most common causes of mixed respiratory acidosis and metabolic

acidosis. DKA is the most common cause for triple acid base disorder. DCLD is the most common cause of mixed metabolic and respiratory alkalosis.

While analyzing individual acid base disorders in combination with both simple as well as mixed, metabolic alkalosis is the most common. It accounts for 49% of cases (14% -simple and 35% mixed). The next common is metabolic acidosis which is seen in 48% (19% simple, 29% mixed). Respiratory alkalosis is seen in 35% of cases (4% simple and 31 % mixed). Respiratory acidosis is the least common acid base disorder seen in 32% (3% simple and 29% mixed).

While analyzing pH and survival rate our study is well correlated with any other studies. Of the total 100 patients 32 patients had expired accounting for a mortality rate of 32%. Of which 10 of them had pH less than 7.2; 1 had pH more than 7.6 and 21 were between 7.2 and 7.6. Of the total cases of 13 who fall under pH less than 7.2 , the mortality rate was 76%. 1 patient had pH more than 7.6 and expired, accounting for the mortality rate of 100%. Among the 86 cases, pH between 7.2 and 7.6, 21 patients expired with mortality rate of 24%.

Irrespective of primary pathology it is the severe acidemia and alkalemia that largely determines the patient status and prognosis (44). Our study is consistent with the statement.

Among the respiratory failure COPD leads the tally with 9 out of 9 patients presented with type II respiratory failure. 46% of patients with sepsis had respiratory failure with type I being 4 and type II being 2. 9 out of 17 patients admitted with OPC poisoning had respiratory failure with majority being type II. 60% of patients with snake bite envenomation had respiratory failure with type I being 1 and type II being 2. 50% of the patients with scorpion sting had respiratory failure with type I being 1, type II being 2. In the miscellaneous group 4 patient had respiratory failure with type I being 4 and type II being 4. Of 14 cases with type I failure 9 had simple and 5 had mixed acid base disorder. Of 28 cases with type II respiratory failure 20 had mixed and 8 had simple acid base disorder. Among the 58 cases of non respiratory failure 35 had mixed and 23 had simple acid base disorder.

Conclusion

CONCLUSION

1. OPC POISONING, SEPSIS, DKA, CKD, COPD are the most common causes of acid base disorders in intensive medical care unit and toxicology unit together accounting for 59% of cases.
2. The most common acid base disorder observed was mixed type, which accounted for 60%, while simple acid base disorder was observed in 40 % of patients.
3. The most common mixed acid base disorder was metabolic alkalosis + respiratory alkalosis which was seen in 22% of cases. 19% of cases had simple metabolic acidosis.
4. Respiratory failure was present in 42% of cases, Type I – 14% and Type II – 28%.
5. OPC poisoning was the most common cause for acid base disturbance in our study (17 %). The most common acid base disorder in OPC poisoning was simple metabolic alkalosis. Respiratory failure was present in 52 % (Type I – 2, Type II – 7)
6. Sepsis accounts for 13 % of cases. The most common acid base disorder in sepsis was mixed metabolic alkalosis and respiratory alkalosis. Respiratory failure was present in 46 % (Type I – 4 , Type II – 2).

7. DKA accounts for 10 % of acid base disturbance. The most common single acid base disorder in DKA was metabolic acidosis.
8. CKD accounts for 10 % of acid base disturbance. The most common acid base disorder in CKD was metabolic acidosis. 7 out of 10 patients expired with mortality rate of 70 %.
9. COPD accounts for 9 % of acid base disturbance. The most common acid base disorder in COPD was mixed metabolic acidosis and respiratory acidosis. All the patients with COPD were admitted with Type II respiratory failure.
10. Among the individual acid base disturbance either in single or mixed form, the metabolic alkalosis is most commonly seen in 49% of cases.
11. 86 % of patients had pH between 7.2 to 7.6, 13 % of patients had pH less than 7.2, 1% of patients had pH more than 7.6 .
12. The mortality and morbidity was more severe in extreme acidemia and alkalemia. In those with pH less than 7.2, the mortality rate was 76 % and in those with pH more than 7.6 mortality rate was 100%, those between 7.2 to 7.6 the mortality rate was 24%.

Bibliography

BIBLIOGRAPHY

1. Gunnerson Kyle J, *Critical Care* 2005; 9 : 508-516.

2. Thomas D.DuBose, Jr. Acidosis and Alkalosis. *Harrison's principles of Internal Medicine.* Vol 1, 16th edn, 2005; 42:263.

3. Eduardo Benchimol Saad, Acid Base Disorders in critical care. *Intensive Care*, 2007;6: 1 – 6.

4. Kassiver et al, Rapid Estimate of Plasma Carbondioxide from pH, *NEJM* 1965; 272:1067.

5. Brady Nilux, Therapy in Nephrology and Hypertension. *W & B Saundas* 1999 ; 1: 292.

6. Kellum JA, Determinants of blood pH in health and disease. *Critical Care* 2000; 4 : 6 – 14.

7. William Kachny, *Manual for Nephrology* , 2003 ; 15 : 49

8. Statement on Acid Base Terminology , *Ann.Inte.med* 1965; 63 : 885.

9. Margaret Bia, *North American Clinics on Acid Base Disease*. 1981; 65 : 347

10. McCurdy D. Mixed Acid Base Disturbance, Diagnosis and Treatment. *Chest* 1971; 62:353

11. Epstein sk et al, Respiratory Acidosis. *Respir Care* 2001 ; 46 : 363.

12. Mitchell et al. The effect of Acid Base on Cardio vascular findings. *Kidney Internat.* 1972 ; 1 :375

13. Kilbun K.H. , Seizures, coma with Alkalosis. Ann.Inter.Med.1966 ; 65 : 979.

14. Madias NE, Adrogue HJ, Acid Base Disturbance in Pulmonary Medicine. *Fluid Electrolyte Acid Base Disorders* 2nd edn, Churchill Livingston 1995 ; 223 :53.

15. Beer R, Goldstein, Effect of Metabolic Alkalosis in Respiratory function. *Can.Med.Ass.J* 1977 ; 117 : 900.

16. Miller PD. Acute Met.Alkalosis precipitating hypercarbia. *JAMA* 1997 ; 238 : 2400.

17. Adrogue HJ, Madias, Management of life threatening Acid Base Disorders. *NEJM* 1998 : 338 :107 – 112.

18. Orchard CH et al , Effects of pH on Contractile function of Cardiac mucscle. *Am.J.Physio* 1990 ; 258 :81 – 90

19. Orchard CH et al, Acidosis and Arrythmias in cardiac muscle. *Cardivas.Res* 1994 ; 28 : 1312 – 19.

20. England BK et al. Abnormalities in protein synthesis and degradation in pH changes. *Am.J.Physio*. 1991 ; 260 : 277 – 282.

21. Hood VL et al. Acid Base homeostasis in keto and lactic acidosis. *Diabetes Rev*. 1994 ; 2 : 177 – 94.

22. Harrington JT, Metabolic alaklosis, *Acid Base* , Boston - little brown, 1987 ; 227 : 306.

23. Rimmer JM, Metabolic Alkalosis. *J. Int.Car.Med* 1987 ; 2 : 137 - 150

24. Fishman, Pulmonary Diseases and Acid Base Disorders, *Manual of Respiratory Medicine*. 3rd edn 1998 ; 2 : 2525 – 27.

25. Ebistein SK et al. Overview of respiratory muscle function. *Clin.Chest.Med* 1994 ; 15 : 619 – 29.

26. Kook W. ABG and pH during sleep in COPD. *Am.J.Med* 1975 ; 58 : 633 – 670.

27. Rebuck AS. Measurement of Ventilatory response to hypercapnia and hypoxia. *Lung Biopsy in Health and Disease*. 1981 ; 2 : 745 – 94.

28. Lane BJ et al. Relation between airway obstruction and CO2 retention in Obstructive airway disease. *BMJ* 1968 ; 3 : 707 – 9.

29. Patrick JM, Influence of Age, Sex, Body, Size and Lung size on control and pattern of breathing in Caucasians. *Resp.Physiol*. 1972 ; 69 : 337 – 50.

30. Schiffman. *Acid Base Disorders in COPD*. Vol 1, 1988 ; 20 :181.

31. Jacobson HR , Seldin DW. On the generation , maintenance and correction of metabolic alkalosis. *Am J Physiol* 1983 ; 245 : 425 – 32.

32. Vernon DD, Gleich MC, Poisoning and Drug overdose. *Critical care clinics* 1997 ; 3 : 647 – 67.

33.Filley GF et al. Acid Regulation and CO2 retention. *Chest* 1970 ; 58 : 417 – 22.

34. Madias et al Hypoxia and Hypercapnia in Asthma. *NEJM* 1968 ; 278 : 1068.

35. Schrier et al. *Diseases of Kidney* 6th edn, Churchill Livingston 2007 ; 3: 2525 -39.

36. Adro et al. Change in Acid Base Pattern in DKA. *NEJM* 1982 ; 307 :1603 – 10.

37. Levovitz HE. Diabetic ketoacidosis. *Lancet* 1995 ; 345 : 767 – 72.

38. Jonathan Cohen, *Infectious Diseases* 2nd edn Mosby 2004 ; 2 : 619 – 22.

39. Lunburg JS et al Septic shock. Analysis of outcome for patients in ward vs Intensive care. *Crit.Care.Med* 1998 ; 26 : 1020 – 24.

40. Narins et al. *The Renal Acidosis issues in Nephrology*. Churchill Livingston 1978 : 30 : 64.

41. Anderson et al. Alkalemia associated morbidity and mortality in medical ward. *South Med.J* 1987 ; 80 : 729 -33.

42. Madias NE et al, Acid Base Disturbance in pulmonary medicine. *Fluid and Electrolyte Acid Base Disorders* 2nd edn Churchill Livingston 1995 ; 223 : 53.

43. Dobes M. Disorders of Acid Base Equillibrium in delirium tremens. *Cas Lek cesk* Mar 8 1993 ; 132 : 142 – 5.

44. Adrogue et al. Life Threatening Acid Base Disorders, *NEJM* 1998 ; 338 : 107.

45. Dhadke V N , Kulkarni P M. The Clinical profile of organophophorous compound poisoning. *JAPI* 2001 ; 49 : 177

46. Bana A K, Bhaskar Y, Clinical correlation of Arterial blood gas Analysis in Organophosphorous compound poisoning. *JAPI* 2001 ; 49 : 180

47. Proudfoot A T, Vale J A Pesticides. *Oxford Text book of Medicine*, vol 1, 3rd edn, 1996 ; 8.3.8 : 1122

48. Garrard C, Foex P Adult respiratory distress syndrome *Oxford Text book of Medicine*, vol 2, 3rd edn, 1996 ; 17.10.20 : 2852 - 60

49. Murphy P A, Septicemia. *Oxford Text book of Medicine*, vol 1, 3rd edn, 1996 ; 7.19.2 : 1020 – 25.

50. Brochard L, Mancebo J et al, Non Invasive ventilation for acute exacerbation of COPD. *NEJM* 1995 ; 333 :817 – 22.

51. Bott J , Caroll M P et al, Randomised controlled trial of nasal ventilation in acute ventilatory failure due to COPD. *Lancet* 1993 ; 341 : 1555 – 7

52. Cohen R D, Woods H F, Disturbances of acid base homeostasis. *Oxford Text book of Medicine*, vol 2, 3rd edn, 1996 ; 11.4 : 1542 - 43

PROFORMA

Sl.No.

Name : Age: Sex :

Clinical Features :

Past H/o : DM / HT / COPD / IHD / CKD / DCLD

Drug H/o :

Diagnosis :

ABG :

pH	PCO_2	PO_2	HCO_3^-	Na^+	K^+	Cl^-

Blood Sugar :

RFT :

Urea	S.Creatinine

LFT :

S.Bilirubin	SGOT	SGPT	S.Proteins	SAP

ECG :

Chest X Ray :

Comments :

MASTER CHART

SL. NO.	NAME	AGE	SEX	DIAGNOSIS	pH	PCO_2	PO_2	HCO_3^-	Na^+	K^+	CL^-	
1.	SANCHIAH	50	M	COPD	7.42	98.3	22.1	30	131	3.2	96	T
2.	MAHENDRAN	25	M	SEPSIS	7.54	28.2	64.4	23.8	134	2.53	98	T
3.	THIRUNIRAISELVAM	58	M	CKD	7.39	26.3	87.5	15.6	139	5.8	140	
4.	RENUKA	20	F	DCLD	7.52	39	83	31.5	136	2.7	116	
5.	GANAPATHY	77	M	CVA	7.54	40.5	71.6	33.1	133	2.3	99	T
6.	MURALI	29	M	CVA	7.44	39.9	99.7	26.7	142	3.9	100	
7.	RADHA KRISHNAN	60	M	MYASTHENIA	7.24	29.2	100	12.4	138	3.5	104	
8.	MOOBINA	20	F	APS	7.50	41.4	100	31.8	137	3.6	84	
9.	PREETHI	20	F	PNEUMOTHOR	7.26	39.3	98	17.5	140	3.5	98	
10.	SARALA	38	F	CCF	7.40	57	63	34	134	3.4	98	T
11.	SHANMUGAM	46	M	PNEUMONIA	7.56	19.1	100	17.0	137	3.8	108	
12.	MUTHU	34	M	DKA	7.30	34.9	102	17.1	132	3.4	117	
13.	FAZIL	39	M	DKA	7.27	15.6	100	7.0	134	3.0	123	
14.	SUNDARI	25	F	HYPOKALEMIA	7.38	50	92	29.2	134	2.1	100	T
15.	GANANASIDDHAN	23	M	OPC POISON	7.33	58	100	30.5	137	4.2	98	T
16.	PERUMAL	40	M	OPC POISON	7.47	48.1	97.8	34.8	137	3.6	119	T
17.	VINESH	42	M	OPC POISON	7.38	55.3	94.7	32.2	132	3.6	103	T
18.	SATISH KUMAR	27	M	ALCOHOLIC	7.50	28.0	95	22.6	132	3.9	104	

SL. NO.	NAME	AGE	SEX	DIAGNOSIS	pH	PCO_2	PO_2	HCO_3^-	Na^+	K^+	CL^-	FA
19	VELU	52	M	ALCOHOLIC	7.50	28.6	90	21.7	136	3.8	110	
20	ETHIRAJ	55	M	SNAKE BITE(NEURO)	7.20	45.1	100	17.4	132	3.4	102	TY
21	RAJA	25	M	SCORPION	6.94	44.3	100	9.5	131	3.6	104	
22	SATYA	22	F	CORROSIVE	7.49	30.5	68.9	23.1	131	2.3	98	TY
23	ANGAIAH	37	M	ODVANTHAL	7.0	55.7	100	13.7	141	1.8	106	TY
24	ANAND	24	M	ATT.HANGING	7.28	43.1	95	20.2	128	34	98	
25	KALPANA	28	F	SEDATIVE	7.43	45	84.3	29.3	140	3.9	96	
26	KANNAYARAM	55	M	COPD	7.22	87.7	27.1	30	135	3.6	98	TY
27	SUBRAMANI	54	M	COPD	7.18	169.4	47.5	32	136	3.8	99	TY
28	NATARAJAN	84	M	SEPSIS	7.58	38.8	100	36.3	134	4.8	98	
29	SEKAR	57	M	SEPSIS	7.31	22.1	100	11.0	147	36	77	
30.	FASHIYA	15	F	CKD	7.52	21.7	100	17.3	138	3.9	112	
31.	ANANDHAN	35	M	CKD	7.23	19.3	91.3	7.9	133	4.5	107	
32.	JOSEPH	45	M	DCLD	7.41	51.1	80	31.8	133	3.2	104	
33.	DAYAKUMAR	45	M	DCLD	7.54	31.6	92.4	26.9	136	4.0	88	
34.	SEKAR	52	M	CVA	7.54	38.6	67.1	32.6	130	3.2	112	TY
35.	THIRUPATHIAH	16	M	PNEUMOTHER	7.50	42.8	100	33	134	3.1	115	
36.	SEKAR	57	M	DKA	7.31	22.1	100	11.0	147	3.6	77	

SL. NO.	NAME	AGE	SEX	DIAGNOSIS	pH	PCO_2	PO_2	HCO_3^-	Na^+	K^+	CL^-	RESP. FAILURE
37.	NOORJAHAN	55	F	DKA	7.32	32	100	16.4	137	3.9	107	
38.	LATHA	32	F	OPC POISON	6.70	84.9	99	10.5	131	4.2	120	TYPE 2
39.	KARUNAN	40	M	OPC POISON	7.48	47.4	100	35.1	134	3.9	106	TYPE 2
40.	VIJAYA	45	F	OPC POISON	7.49	38.7	92	28.9	139	3.6	100	
41.	KANNAN	41	M	OPC POISON	7.58	22	100	32	136	2.9	111	
42.	SUNDARA RAJAN	45	M	ALCOHOLIC	7.54	15.8	90	30.1	132	3.1	106	
43.	SUNDARAM	40	M	ALCOHOLIC	7.54	35.8	100	31.1	130	3.1	100	
44.	ANNA DURAI	34	M	SNAKE BITE (VASCULO TOXIC)	7.50	35	94	26	124	3.8	106	
45.	DORAIKANNAN	52	M	SNAKE BITE	7.41	31	73.4	19.6	144	3.6	109	TYPE 1
46.	SELVI	35	F	CORROSIVE	7.22	23.2	96.2	10.9	150	3.8	105	
47.	SWAPNA	23	F	CORROSIVE	7.29	56.6	51.7	26.9	123	3.6	107	TYPE 2
48..	VENGATESH	25	M	CORROSIVE	7.33	40.7	90	21.3	145	3.1	106	
49.	PRABHU	23	M	HANGING	7.49	42.1	90.6	31.5	139	3.3	105	
50.	KAJA MOHIDEEN	65	M	DKA	7.23	28	95.9	7	146	5.2	106	
51.	AMMU	20	F	DKA	7.38	22.5	90	13.3	136	3.6	98	
52.	KASI	40	M	COPD	7.05	126.1	86.9	24.2	138	3.9	100	TYPE 2
53.	KOUSALYA	63	F	COPD	7.44	86.5	46.6	58	135	3.1	89	TYPE 2
54.	JAMUNA	19	F	SEPSIS	7.32	32.1	52.3	14.2	134	3.2	116	TYPE 1

SL. NO.	NAME	AGE	SEX	DIAGNOSIS	pH	PCO_2	PO_2	HCO_3^-	Na^+	K^+	CL^-	RESP. FAILURE
55.	SUBRAMANI	39	M	CKD	7.50	15.7	93	12.0	119	4.6	104	TYPE 2
56.	KATHIRVEL	16	M	DCLD	7.54	38.5	77.3	32.7	130	2.9	1.9	TYPE 1
57.	VIYAY	42	M	OPC POISON	7.54	60.2	76	36.5	134	3.6	111	TYPE 2
58.	SUNDARA RAJA	46	M	ALCOHOLIC	7.24	34.4	94.4	14.7	137	3.9	124	
59.	ATHANKARAIYAN	60	M	ALCOHOLIC	7.44	60.1	100	40.8	131	2.6	89	TYPE 12
60.	AKTCHAYA	24	M	SNAKEBITE	7.17	80	100	17.9	137	3.6	105	TYPE 2
61.	RAMA	25	F	SCORPION	7.40	22.2	58.9	13.5	132	3.2	98	TYPE 1
62.	SUMATHI	26	F	CORROSIVE	7.49	3.5	67.7	23.1	140	4	98	TYPE 1
63.	NITHIYA	20	F	HANGING	7.58	34.2	90	31.4	143	3.1	111	
64.	VINCENT	23	M	HANGING	7.38	36.1	98	21.3	135	3.4	105	TYPE 2
65.	VELU	28	M	DKA	7.42	28.8	92.6	15	136	3.7	98	
66.	KANDAVEL	17	M	DKA	7.42	28.8	98	15	135	3.6	978	
67.	SHANTHA	62	F	COPD	7.34	138.9	79.2	59.1	123	3.2	95	TYPE 2
68.	VENKADESHAN	34	M	SEPSIS	7.48	37.8	93.6	27.6	140	3.6	111	
69.	LEELA	55	F	SEPSIS	7.43	50.9	92	33.2	134	3.1	108	TYPE 2
70.	MOHANA	58	F	CKD	7.58	33.2	86.8	30.4	125	4.6	73	TYPE 2
71.	SATHIYANARAYAN	72	M	OPC POISON	7.1	15.0	60	12	136	3.8	96	TYPE 1
72.	CHINNAKUTTI	32	M	OPC POISON	7.46	34	100	23	139	3.2	108	

SL. NO.	NAME	AGE	SEX	DIAGNOSIS	pH	PCO_2	PO_2	HCO_3^-	Na^+	K^+	CL^-	RESP. FAILURE
73	KASTURI	50	F	OPC POISON	7.53	35.7	100	29.7	135	3.6	91	
74	MARTIN RAJ	24	M	SCORPION	6.95	70.7	35.8	15.3	154	2.9	126	TYPE 2
75	RENUKA	32	F	SNAKE BITE	7.59	28.3	100	26.7	135	4.4	101	
76	THANGAM	30	F	DKA	7.43	24.3	98	15.9	136	3.5	100	
77	CHELLIAH	77	M	COPD	7.51	49.4	88.9	58.7	137	4.1	98	TYPE2
78	PERIASAMY	60	M	COPD	7.20	97.2	100	37.9	133	4	108	TYPE2
79	SIDHIKA	45	F	SEPSIS	7.62	38.1	49.5	36.8	121	2.7	101	TYPE1
80	KANTHAMANI	45	F	SEPSIS	7.52	38	83.2	30.9	132	3.3	109	
81	MOHANA	55	F	SEPSIS	7.46	50.9	83	35.5	134	3.7	105	TYPE2
82	INDHUMATHI	29	F	CKD	7.08	31.1	100	9.2	137	5.8	100	
83	DHANASHEKAR	50	M	CKD	7.30	49.3	100	24.0	150	6.46	115	
84	SUMATHI	22	F	OPC POISON	7.49	30.5	87.7	23.1	137	3.28	103	
85	RAMKUMAR	45	M	OPC POISON	7.46	34	100	24	144	3.3	101	
86	LAKSHMI	20	F	SCORPION	7.17	45.2	90	16.2	136	3.2	98	TYPE2
87	SHANKAR	24	M	SCORPION	7.23	28	94	7	138	3.6	99	
88	RAMU	35	M	DKA	7.40	28.6	95	17.5	147	3.5	98	
89	RAJAN	60	M	COPD	7.31	69.7	95	28.5	132	3.2	85	TYPE2
90	NOORJAHAN	55	F	SEPSIS	7.32	32.3	100	16.4	137	3.9	107	

SL. NO.	NAME	AGE	SEX	DIAGNOSIS	pH	PCO_2	PO_2	HCO_3^-	Na^+	K^+	CL^-	F
91.	AMALANATHAN	45	M	SEPSIS	7.51	41.4	79	23	133	3.4	102	T
92.	SUNDARI	60	F	CKD	7.32	24.0	67	12.1	139	4.6	105	T
93.	PAGALESWARI	32	F	SEPSIS	7.54	44.2	96	37.6	137	4.0	112	
94.	RAFIQ AHMED	62	M	CKD	7.44	31.3	100	20.8	134	4.5	94	
95.	PARVATHI	23	F	OPCPOISON	7.30	32.5	85	15.8	130	4.12	109	T
96.	VINAYAGAM	30	M	OPCPOISON	7.09	39	100	11.6	135	3.8	102	
97.	SUBRAMANI	50	M	SEPSIS	3.38	34.9	90.6	20.3	134	3.8	108	
98.	DEVAMMAL	40	F	CKD	7.10	7.0	98	2.1	136	4.7	103	
99.	KARUNAKARAN	41	M	OPCPOISON	6.93	106	58	22.1	142	4.5	104	T
100	RATHINAMMAL	55	F	OPCPOISON	7.34	30.8	100	16.5	140	3.6	105	